Commentary

The San Francisco Bay Epidemiology Studies on Asbestos in Drinking Water and Cancer Incidence: Relationship to Studies in Other Locations and Pointers for Further Research

by Marty S. Kanarek*

I would like to give my views on why the results in the San Francisco Bay studies (1,2) are generally more positive than the other ecological epidemiology studies that relate asbestos in drinking water to the incidence of cancer. Many other people have speculated, and I think it would be very appropriate here for me to do this also.

I see four possible explanations for the generally larger number of positive results from the San Francisco Bay studies as compared with the studies in Puget Sound (3), Connecticut (4, 5), and other places.

First, it is possible that the San Francisco Bay Area results are spurious or that the epidemiology studies done in other areas lack enough sensitivity to detect a real effect. This seems doubtful, however, considering how much effort has been made by various parties to negate the San Francisco positive results and to no avail. Also, the studies from Duluth (6), Connecticut (4, 5), Puget Sound (3), and San Francisco Bay (1, 2) are apparently so well and thoroughly done that it is hard to dismiss any of them as insensitive in design.

The second explanation is one of simple statistical power considerations. The San Francisco Bay Area study used the largest population. Thus, for cancers of low incidence, it had enough power to detect an association, whereas the other studies had too few cases to merit a similar association.

The third explanation concerns differences in fiber amounts, fiber size and the follow-up period.

West Coast studies could be that longer fibers were absent in Puget Sound as compared to the distribution of fiber lengths in the Bay Area. Likewise, the asbestos counts in Connecticut were low compared with those in the San Francisco Bay Area. As for follow-up, only now have enough years passed in Duluth to approach encompassing the latent period necessary.

The fourth explanation concerns the biological

An explanation for the differences found in the

The fourth explanation concerns the biological mechanism of asbestos-related carcinogenesis. The San Francisco Bay Area population may be exposed to some cocarcinogen or not exposed to a possible protective factor. This would especially be important if asbestos were not an initiator. Neither the initiator in the San Francisco Area nor the protective factor in the other areas would have to be in drinking water—food would also be a prime candidate.

Practically, what should be our next steps in answering our questions concerning asbestos in drinking water and cancer? Clearly, population-based incidence case-control studies should now be done on stomach, pancreas, and peritoneal cancers because of the findings of all the ecologic studies put together. Remember, a misdiagnosed peritoneal mesothelioma would most likely be called a pancreatic cancer.

In proper epidemiologic progression, we have conducted descriptive studies in order to refine our hypothesis, in this case to select which cancer body sites warrant case-control studies. Incidence, not mortality case-control studies, should now be done because of the complicated exposure histories necessary in this instance to do a proper study. For individuals with newly diagnosed cancer of the pancreas, peritoneum, and stomach,

^{*}Department of Preventive Medicine and The Institute for Environmental Studies, 504 N. Walnut St., University of Wisconsin, Madison, WI 53705.

and for the appropriate control cases, such incidence case-control studies would seek detailed lifestyle and exposure histories, including lifetime residence histories, occupation, drinking water consumption habits, diet (specifically vitamin intake), cigarette smoking and other factors.

An incidence case-control study would be informative concerning any potential etiologic variables or combination of variables on which data could be gathered from cases and controls. Fiber length should be incorporated in the assessment of individual lifetime asbestos ingestion. Given the basic positive results—the presence of longer fibers and the speculation that a cocarcinogen exists or that an inhibiting or detoxifying variable is absent—the San Francisco Bay Area would be a most fruitful location for such a study.

The views and policies presented by the author in this commentary do not necessarily reflect those of the U.S. Environmental Protection Agency. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

REFERENCES

- Kanarek, M. S., Conforti, P. M., Jackson, L., Cooper, R. C., and Murchio, J. C. Asbestos in drinking water and cancer incidence in The San Francisco Bay area. Am. J. Epidemiol. 112: 54-72 (1980).
- Conforti, P. M., Kanarek, M. S., Jackson, P. M., Cooper, R. C., and Murchio, J. C. Asbestos in drinking water and cancer in the San Francisco Bay Area: 1969-1974 incidence. J. Chronic Dis. 34: 211-224 (1981).
- Polissar, L., Severson, R. K., Boatman, E. S., and Thomas, D. B. Cancer incidence in relation to asbestos in drinking water in the Puget Sound region. Am. J. Epidemiol. 116: 314-328 (1982).
- Harrington, J. M., Craun, G. F., Meigs, J. W., Landrigan, P. J., Flannery, J. T., and Woodhull, R. S. An investigation of the use of asbestos cement pipe for public water supply and the incidence of gastrointestinal cancer in Connecticut, 1935–1973. Am. J. Epidemiol. 107: 96–103 (1978).
- Meigs, J. W., Walter, S. D., Millette, J. R., Craun, G. F., Heston, J. F., Woodhull, R. S., and Flannery, J. T. Asbestos cement pipe and cancer in Connecticut, 1955–1974. J. Environ. Health 42: 187–191 (1980).
- Sigurdson, E. E., Levy, B. S., Mandel, J., McHugh, R., Michienzi, L. J., Jaggar, H., and Pearson, J. Cancer morbidity investigation: lesson from the Duluth study of possible effects of asbestos in drinking water. Environ. Res. 25: 50-65 (1981).